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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN PITCH LANDON, M.D., Editor

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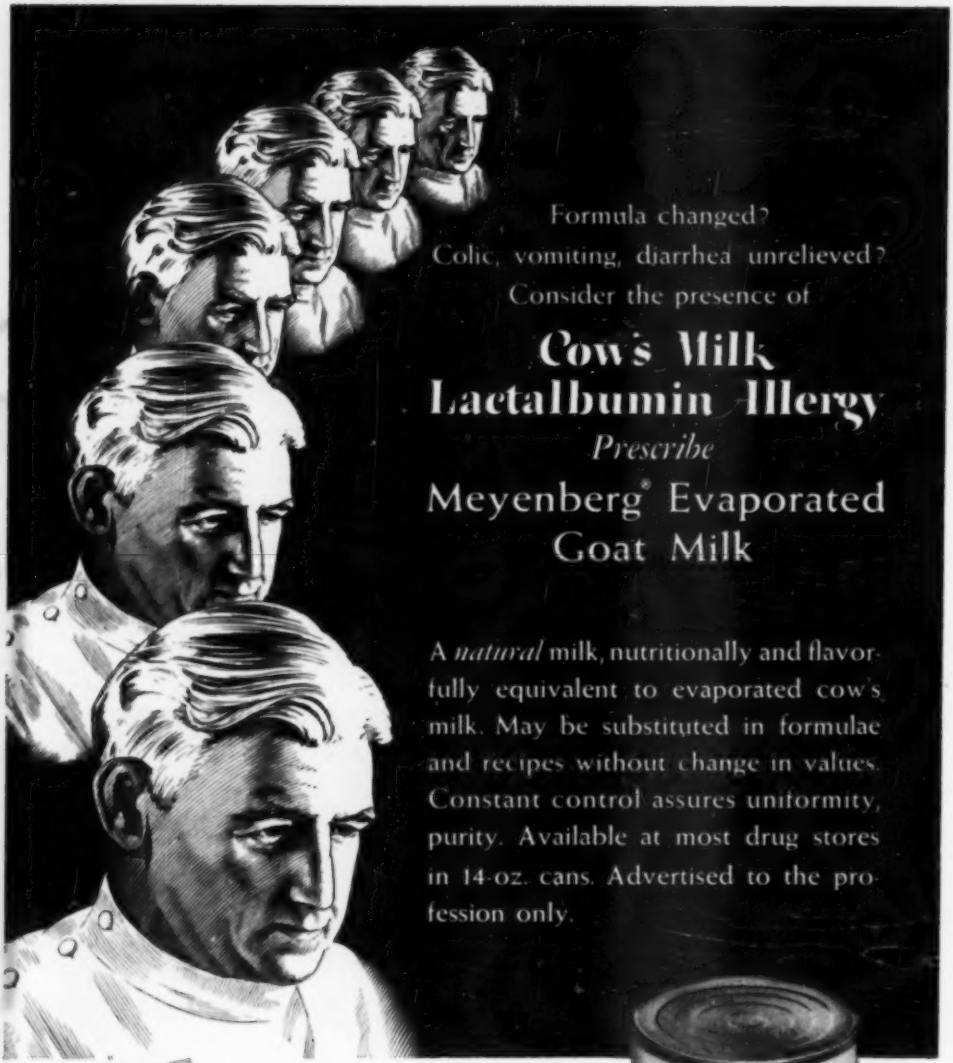

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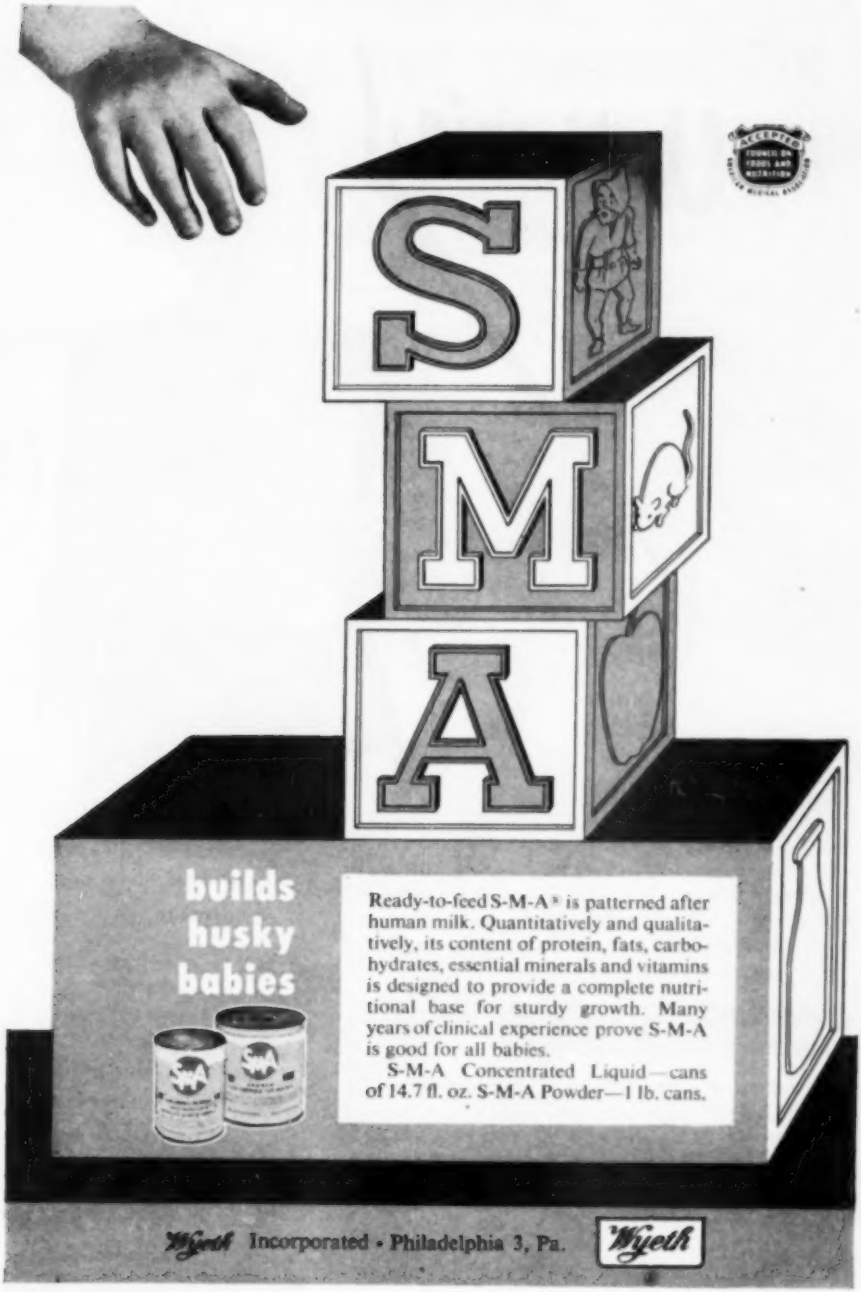
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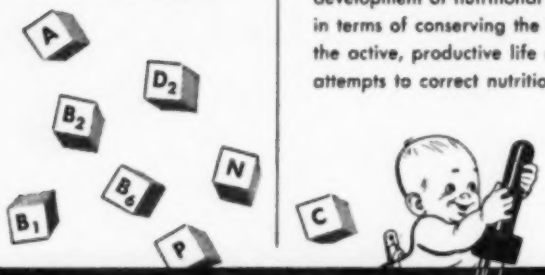

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1. Wolfe, S. O.: *Med. Clin. North America*, 33:1709, Nov., 1949.

2. Goodhart, R. S.: *Postgrad. Med.*, 5:191, Mar., 1949.

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* Vital Statistics—Special Reports: Vol. 25, No. 12, National Office of Vital Statistics, Washington, D. C. (Oct. 15) 1946, p. 206.

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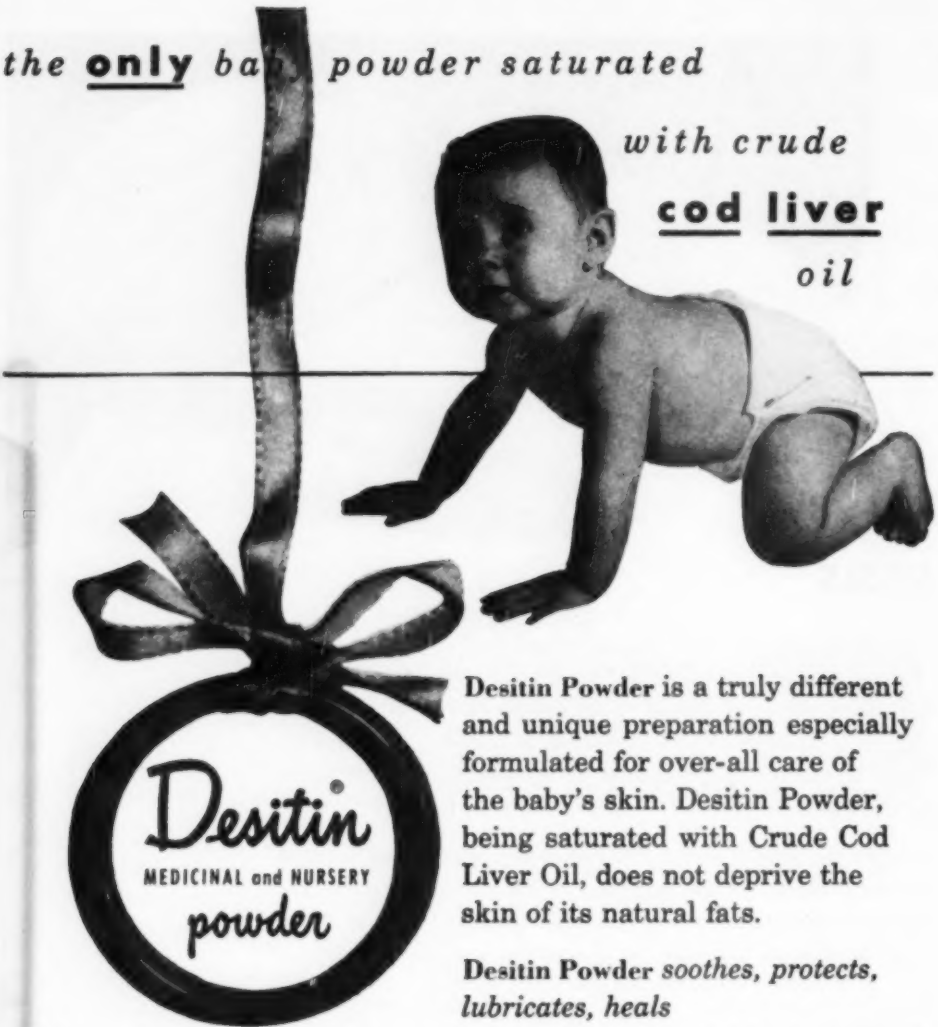

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OMENTAL SEGMENTAL INFARCTION IN INFANCY

REPORT OF CASE

DANIEL H. MANFREDI, M.D.
New York.

Among the rare clinical entities simulating acute appendicitis in children, none is so elusive of diagnosis as segmental omental infarction. The diagnosis is always made after the peritoneal cavity is opened. In the literature this condition has been classified as primary epiploitis, segmental infarction and torsion of the omentum. The lesion is always vascular in nature, being either embolic or thrombotic. This pathological entity is extremely rare in children with hardly a case having been reported in the last twenty years. There have been many cases reported in adults, especially in those having a concomitant generalized vascular disturbance such as Buerger's disease, arteriosclerosis, cardiac disease or hypertension. Totten's theory[1] states that a thin walled omental vessel, engorged following a meal, when subjected to a strain causes rupture and hemorrhage with infarction. The following child was recently seen on the wards of Knickerbocker Hospital in New York.

CASE REPORT

A nine-year-old female child was admitted to the hospital with a history which was heralded by generalized abdominal pains and nausea twenty-four hours prior to entering the hospital. The pain became progressively worse and shifted to the right lower quadrant where it remained. There was no history of diarrhea, upper respiratory disease or previous operations. The child recalled no re-

cent trauma to her abdomen. Physical examination revealed a well-developed female with pain in her abdomen. Her temperature was 100.6° F., pulse 90 per minute, respirations 20 per minute. The white blood count was 13,800 with 78 per cent polys, 16 per cent lymphocytes, 6 per cent monocytes. Urine analysis was essentially normal. Hemoglobin was 13.5 grams and the red blood cell count was 4.2 million. The only abnormal physical findings were in her abdomen which was moderately distended. Throughout the abdomen there was generalized tenderness most marked in the right lower quadrant with some rebound tenderness over McBurney's point. A diagnosis of acute appendicitis was made and three hours post-admission a laparotomy was performed through a McBurney type of incision. There was a small amount of clear serous fluid in the peritoneal cavity. The appendix appeared normal and was removed in routine manner. Exploration of the pelvis and ileum failed to reveal any pathology. However, in the right lower outer angle of the omentum there was found a tumor-like mass measuring about 2 by 3 centimeters. This mass was well circumscribed and indurated with the surrounding omentum appearing perfectly normal. This mass with a margin of omentum was removed by a clamp and ligature technique. Cultures were taken of the peritoneal fluid and the abdomen was closed in layers without drainage. The child experienced a smooth convalescence, the wound healing per primum, and she was discharged home eight days post-admission in excellent condition.

The pathological report was as follows: Normal appendix; omental tumor caused by previous twisting and infarction of omentum and producing the clinical picture. Tumor shows on section a red meaty cut surface with some thick walled vessels.

SUMMARY

A rare clinical entity is presented which closely simulates acute appendicitis and from which it is most difficult to differentiate. This is especially true in infancy where a concomitant blood vessel disease is a rarity. The treatment advised is laparotomy with excision of the mass. Search of the literature fails to unfold cases occurring in infants although there are numerous cases reported occurring in adults. The probable explanation of the above entity is due either to twisting of the omentum, which is always thin and

poorly developed in children, or to a direct trauma causing infarction.

REFERENCE

1. Totten, H. P.: Primary Odiopathic Segmental Infarction of the Greater Omentum. Am. J. Surg., 56, 676-679, 1942.

642 *Park Avenue.*

SURGICAL TREATMENT OF HODGKIN'S DISEASE. (Semaine des Hôpitaux de Paris, 25: 3503, Nov. 14, 1949). Bernard and Ossipovski operated on 3 patients with Hodgkin's disease, 2 boys aged 11 and 9 years and a young woman aged 19, removing a cervical lymph node in the first patient and a subclavicular lymph node in the second and third patients. The patients were alive and in good health, eleven, eight and six years, respectively, after the intervention. Surgical treatment is indicated in recent, isolated lesions of moderate size. It is contraindicated in multiple lymph node or visceral involvement associated with fever, although even then results may not be unfavorable. Clinical, experimental and therapeutic data show that a malignant disease of the blood may be localized in its early stage and that early surgical removal of the initial focus may result in long survival. According to the author's concept, the nature of malignant diseases of the blood may differ. Each disease may be formed by a combination of various syndromes, some resulting in two or three foci in the initial stage and some in a single focus in the initial stage. The occurrence of a single lesion in the initial stage seems to be demonstrated not only in Hodgkin's disease but in other malignant diseases of the blood as well. This concept makes it imperative to track down the initial and localized manifestation. Removal of the initial tumor, whether in lymph nodes, skin, bone or viscera, rather than the generally practiced biopsy, is advisable in the absence of contraindications. Results may not be brilliant and often may be disappointing. The initial lesion in Hodgkin's disease may be mediastinal or abdominal and therefore inaccessible. The superficial adenopathy may appear isolated, but careful examination may reveal an affected mediastinal lymph node, contraindicating surgical intervention. The results obtained in the authors' cases justify cautious pursuit of surgical therapeutic trial.—*Journal A.M.A.*

BULBAR POLIOMYELITIS*

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The purpose of this paper is to review the clinical aspects of bulbar poliomyelitis with emphasis on present day concepts. The importance of this subject lies in the fact that bulbar involvement produces the most serious form of the disease and accounts for almost the entire mortality attributable to poliomyelitis. The spinal type is not lethal except where there is involvement of the muscles of respiration, and then death may result from either asphyxia, if the respirator is not available, or from pulmonary complications. Bulbar poliomyelitis calls for careful and continuous observation because skillful intervention at the proper time may be lifesaving.

Terminology. The term "bulbar poliomyelitis," as it is commonly used, refers to that form of the disease in which there is involvement of the cranial nerves and the vital centers (respiratory and circulatory) either separately or in combination. To many, "bulbar poliomyelitis" connotes those cases which have palatal, pharyngeal and laryngeal involvement and especially those cases with difficulty in swallowing. The term "bulbar" anatomically refers to the bulb or medulla oblongota, and only the lower cranial nerves, the 9th, 10th, 11th and 12th, arise from cells in the medulla, while the upper cranial nerves have their origin in the pons and the midbrain. To characterize poliomyelitis as either a "pontine type" or "bulbar type" though anatomically correct may be confusing in present-day usage. It may be advisable to continue the all-inclusive term "bulbar poliomyelitis" to denote involvement of the entire brain stem, including the medulla, pons and midbrain.

From the clinical point of view, involvement of the cranial nerves and the vital centers may occur alone, but often there is simultaneous involvement of the spinal nerves, "bulbospinal poliomyelitis," or of the higher brain centers (cerebral) "bulboencephalitic poliomyelitis." These forms are included in this discussion of bulbar poliomyelitis.

Incidence. Bulbar poliomyelitis varies in frequency in different

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epidemics and different years. Differences in reported incidence may be due in part to variations in classification and to the detection of mild cases and their inclusion or omission in the figures. Wickman,[1] in 1905, in his classic report on 1,025 cases of polio-

TABLE 1. *Poliomyelitis, Willard Parker Hospital. Poliomyelitis and Bulbar Cases; Deaths and Case Fatality Rates.*

Year	Total No. Polio. Cases	Total No. Bulbar Cases	Bulbar Cases %	Total Polio. Deaths	Polio. Case Fatality Rate %	Bulbar Deaths	Bulbar Case Fatality Rate %
1933[7]	273	123*	45.0	31	11.3	30	24.4
1935[8]	686	69	10.0	18	2.6	18	26.1
1944	493	112	22.7	31	6.2	28	25.0
1948	203	45	22.1	9	4.4	8	17.7
1949	526	126	23.9	40**	7.6	39***	30.9

*87 of these (32.0 per cent of total polio. cases) had difficulty in swallowing.
**1 spinal respirator case died 33 days after admission.
***1 of these died 4 months after admission.

myelitis in Sweden, noted bulbar involvement in about 6 per cent of the cases. Reports in more recent years[2,3,4,5] give much higher incidence rates ranging from 9 per cent to 23 per cent; and in one report[6] as high as 36 per cent in the patients over 16 years of age. The incidence of bulbar poliomyelitis at the Willard

TABLE 2. *Bulbar Poliomyelitis, Willard Parker Hospital,* 1949. *Sex Distribution, Incidence and Case Fatality Rates by Age Groups.*

Age (Years)	Cases				Deaths			
	Male	Female	Total Cases	Bulbar Cases %	Male	Female	Total	Case Fatality Rate %
0- 4	6	7	13	10.3	2	2	4	30.7
5- 9	33	14	47	37.3	9	5	14	29.7
10-14	19	15	34	27.0	6	1	7	20.5
15-19	10	7	17	13.5	4	4*	8	47.1
20-up	5	10	15	11.9	2	4	6	40.0
Total	73	53	126	100.0	23	16	39	30.9

*1 patient died 4 months after admission (bulbospinal respirator case).

Parker Hospital for 5 separate years is shown in Table 1. It varies from 10 per cent to 45 per cent.

Bulbar poliomyelitis seems to be more prevalent in the male, as is the case with poliomyelitis generally. During 1949 (Table 2) there were 126 bulbar patients treated in the Willard Parker

Hospital, and of these, 73 (57.9 per cent) were males and 53 (42.1 per cent) were females. However, in the age group 15 years and over, there were slightly more females. Of the total poliomyelitis admissions for the year the incidence of bulbar poliomyelitis in the age group under 15 years was 23.8 per cent, and in the group 15 years and over it was 25.2 per cent. The youngest patient was 7 months of age, and the oldest was 33 years, both females.

Pathology. In fatal cases of bulbar poliomyelitis, pathologic lesions may be demonstrated in other parts of the central nervous system—in the cerebral cortex and in the spinal cord—even though there have been no clinical signs to indicate such involvement. It appears too that some bulbar centers are more vulnerable functionally, for although most cranial nerve nuclei in the fatal cases show lesions, yet it is the muscles of the face, pharynx and larynx that are most often involved in bulbar cases, while muscles innervated by the oculomotor, trigeminal and hypoglossal nerves are not so often affected. It may be that from the clinical aspect slight paralyses are overlooked in the severely ill bulbar patient.[9] The lungs in bulbar poliomyelitis often show severe edema, congestion, interstitial or bronchopneumonia[10] and focal atelectasis. In a fair percentage of patients there is found a focal interstitial myocarditis, but whether this heart muscle involvement is due to the virus of poliomyelitis, or is secondary to some infection, usually in the respiratory tract, is not definitely known.[11, 12, 13] In the stomach there may be congestion of the mucosa and submucous hemorrhage; in some cases there is surface erosion of the mucosa.[14] More severe ulceration of the stomach has been reported.[15]

Mortality. Bulbar poliomyelitis accounts directly or indirectly for almost all the poliomyelitis deaths (Table 1). Mortality rates in bulbar poliomyelitis vary in different years, ranging as high as 39 per cent.[7, 16, 17, 18] Mortality is higher in adults than in children.

During the year 1949 there were 40 poliomyelitis deaths at the Willard Parker Hospital, 39 of which had bulbar involvement. Of the bulbar deaths, 23 were males and 16 females; 6 (26.6 per cent) of the males were 15 years of age or older, while 8 (50 per cent) of the females were in this age group (Table 2).

The severity of bulbar poliomyelitis and the uncertainty and rapidity of the course of the disease are illustrated by our mor-

tality statistics for this year. Many cases treated or observed for a time elsewhere, usually at home, were rushed to the hospital in critical condition. Others apparently not so critically ill progressed rapidly and death ensued quickly after admission. Of the 39 bulbar deaths, 15 occurred within 24 hours after admission. Of these 15 deaths, 6 occurred in less than 4 hours, and 3 in a period of from 4 to 8 hours.

Symptomatology. As in other forms of poliomyelitis there are prodromal symptoms, such as headache, fever, malaise, mild sore throat, nausea and vomiting, and constipation, although occasionally there may be diarrhea. The sore throat present early in about 10 per cent or more of the patients may in some cases be the herald of the bulbar form of the disease. It is probably of neurogenic origin, rather than a true pharyngitis, since there is only hyperemia and rarely evidence of inflammation or exudate.[19]

In some cases the initial symptoms subside for a period varying from 1-7 days prior to the onset of central nervous system involvement (the second phase of the biphasic "dromedary" course). Symptoms, such as fever and headache, reappear, and backache, drowsiness and irritability follow, and then very quickly, or within a few days thereafter evidence of bulbar involvement appears. In many instances the prodromata are absent, or mild, and therefore disregarded, and the first evidence of illness is the appearance of symptoms indicative of bulbar involvement.

In our cases of poliomyelitis the biphasic pattern of the disease, which has been noted by some to occur in up to one-third or more of the patients, presented itself much less frequently; this less frequent occurrence may be due to the variation in history taking. Some workers doubt the existence of a biphasic course of poliomyelitis, feeling rather that the first febrile illness is due to some other infection.[20]

Involvements of the upper cranial nerves are important, but they do not in themselves endanger the patient's life. Paralysis of the facial nerve (cranial nerve VII) is the most common, occasionally the eye muscles are affected (III, IV, VI) while disturbances in mastication (V) occur less frequently. The existence of these paralyses should make one consider the likely presence of the more serious involvements of the lower cranial nerves and vital centers. The commonest signs of bulbar poliomyelitis are due to lesions

of the lower cranial nerves, particularly the 9th and 10th. We reported a series of 123 patients with cranial nerve lesions in which difficulty in swallowing occurred in 87.[7] In the series from Minneapolis, of 107 cases of bulbar poliomyelitis in children all but 11 had involvement of the 10th cranial nerve, the vagus.[3] When these nerves are affected there is weakness or paralysis of the soft palate, the pharynx and the vocal cords. When the soft palate and pharynx are involved the patient may have a nasal twang to the voice, accumulation of secretions in the oropharynx and difficulty in swallowing. There may be regurgitation of fluid through the nose, especially in children. Examination will reveal either no movement, or irregular movement of the soft palate, loss of the gag reflex and pharyngeal weakness. When the vocal cords are involved there may be hoarseness and in rare instances loss of voice. Laryngeal stridor, cyanosis and severe inspirational dyspnea may be due to bilateral abductor paralysis of the vocal cords. Direct laryngoscopy will reveal the vocal cords approximated in the midline. Reestablishment of the airway by an endotracheal tube and then tracheotomy can be lifesaving. Spasm of the glottis, usually reflex in nature resulting from attempts to keep secretions out of the larynx and trachea, is likely to respond to less drastic procedures, such as suction and change of position.

Weakness of the sternocleidomastoid and trapezius muscles occurs, but this is not of serious import. Paralysis of the tongue interferes with the movement of food and the expectoration of saliva, and sometimes with breathing.

The weak cough or the inability to cough which contributes to the bulbar patient's difficulty in keeping his airway clear may be due to several causes: weakness or paralysis of the vocal cords; paralysis of the muscles of respiration, especially the diaphragm; central depression of the cough reflex; the accumulation of secretions over the glottis.

When the invasion of the medulla is more extensive and affects not only the cranial nerves but also the vital centers (respiratory and circulatory), the outlook is more grave. Symptoms of damage to these centers usually appear somewhat later in the illness than do the cranial nerve palsies and sometimes may be present without severe cranial nerve involvement. Damage to the respiratory center manifests itself by irregular, shallow, hiccough-like breath-

ing which may go on to periods of apnea and finally cessation of respiration. Some patients may show evidence of circulatory center failure. They have a dusky red appearance with a very rapid, often irregular pulse and a falling blood pressure that may be difficult to maintain with stimulants. Symptoms of terminal shock may appear, and the heart sounds become imperceptible before respiration ceases. Fever, due probably to involvement of the temperature regulating center, may reach unusually high levels in such cases. If the damage to the vital centers is severe, the outcome is fatal despite all efforts.

Hypertension, both systolic and diastolic, may occur during the acute stage of bulbar poliomyelitis. The origin of the hypertension is not clear; dysfunction of the cardiovascular centers and vasomotor disturbances may account for it, and hypoxia (or even hypercapnea) may play some role.[3, 21, 22]

Certain points stand out in the general symptomatology of bulbar poliomyelitis: 1. The bulbar patient is inclined to be more severely ill early in the course of the disease, and the paralyses appear more rapidly than in those with spinal involvement. 2. The paralyses may be slight, sometimes fleeting and of questionable presence, or they may be severe enough to be a threat to life. What appears to be a mild illness, in a few hours may result in death from respiratory and cardiovascular failure, or severe pulmonary complications. 3. Multiple paralyses in the same patient often produce an overlapping of symptomatology, and it may be difficult at times to differentiate the effects of these paralyses.

Hypoxia. Nervous tissue is particularly susceptible to the deficiency of oxygen, possibly more so in adults than in infants and children, and this is especially important in poliomyelitis where the virus has damaged the nerve cells and affected the circulation to the cells.[23] In some instances, true brain tissue changes in poliomyelitis have been attributed to anoxia.[24] The importance of hypoxia in acute poliomyelitis has been emphasized in more recent years as the basis for many of the symptoms and as contributing to the severity of the disease.[4, 16, 23, 25] Many of the so-called "encephalitic-like" symptoms and manifestations of poliomyelitis, such as anxiety, restlessness, hyperexcitability, muscle tremors and twitching, mental confusion, lethargy, coma and convulsions, have been attributed to hypoxia. Although these may be the result of

true polio-encephalitis from inflammatory lesions due to viral action, in many instances they clear up under intensive and proper oxygenation of the patient after clearance of his airway. Hypercapnea (excessive CO_2 in the blood) may contribute to this symptomatology and can be dangerous in itself.[22]

Cyanosis is a late sign of hypoxia and often heralds the death of the patient. Treatment with oxygen should be instituted before there is visible cyanosis, when there are early signs of oxygen deficiency, such as restlessness, apprehension, sleeplessness, increasing pulse rate and respiratory effort. The oximeter[26, 27, 28] is of value in detecting early hypoxic states.

Respiratory Difficulties. The respiratory difficulties that occur in bulbar poliomyelitis and which contribute to the oxygen deficiency and its manifestations result from several causes: 1. Obstruction of the upper airway by secretions, food or vomitus. 2. Spasm of the larynx, or paralysis of the vocal cords. 3. Failure of the central control of breathing (respiratory center), or peripheral failure (muscles of respiration). 4. Pulmonary complications, such as atelectasis, pulmonary edema or pneumonia.

Sometimes it is difficult to determine which of these mechanisms plays the role or the predominating role—for example, whether the breathing irregularities are due to a true involvement of the respiratory center, or due rather to the patient's unwillingness or inability to take a deep breath because of accumulated secretions in the pharynx and the protective action on his part to prevent "drowning in his secretions." In some instances the odd breathing patterns may suggest involvement of the intercostal muscles and diaphragm, but suddenly, or on demand, the patient will take a deep breath which will reveal that there is adequate chest excursion.

Treatment of Bulbar Poliomyelitis. Thus far there is no specific treatment which will alter the course of the disease in the human body. Serums, sulfonamide drugs and the antibiotics have not proved effective. A sulfonamide drug, Darvisul, N-(2-thiazolyl)-phenol sulfonamide, or phenosulfazole was reported to protect mice against a mouse neurotropic virus, and it was recommended for use in the treatment of human poliomyelitis.[29] Early experience with the sulfonamide drugs in acute poliomyelitis indicated that they were of no value experimentally or clinically, and that actually

they might be harmful.[30-33] It may be worth noting our experience at the Willard Parker Hospital with Darvisul.* During 1948, 45 of the 203 poliomyelitis patients admitted had bulbar involvement. In the summer of that year, Darvisul was given to 36 patients, 15 of whom were bulbar cases. There were side effects in 10 of the patients who received the drug: severe nausea, vomiting, extreme restlessness in 5; hematuria in one; eosinophilia in another; 3 had convulsions, one of whom, a bulbar case, died. Paralyses developed in nonparalytic patients and progression of paralysis occurred in others. Of a total of 9 deaths in 1948, 8 were bulbar cases, and 3 of these were in the Darvisul treated group. It was the feeling that the drug was ineffective in human poliomyelitis, and recent reports corroborate this.[34-36]

However, there are certain methods of treatment which will reduce mortality especially if instituted early, or at the proper moment in some situations. These are directed toward the prevention of complications which may occur in bulbar poliomyelitis.

Because of the pooling of material in the mouth and throat due to the inability to swallow or expectorate, and because of the tendency of bulbar patients to vomit, there is the danger of aspiration and attacks of choking and cyanosis. To counteract this, postural drainage is instituted immediately, the foot of the bed being elevated at least one foot and preferably more. The patient is best kept on his side, or on his abdomen with his face turned to one side. Suction of the mouth and throat is very important; it must be done carefully with a minimum of trauma to tissue, and only as often as needed to produce relief. The electric motor type aspirator should provide adequate but not excessive suction, the catheter being of rubber, although some prefer a rigid metal suction tube. Nurses and attendants should be instructed in the proper technique of aspiration. The patient who does not have paralysis of the upper extremities and who is cooperative can be taught to use the apparatus and suction his own mouth and throat.

Patients who show signs of having aspirated secretions, food or vomitus, and this may occur especially if attempts to swallow are urged too soon, may require suction through the laryngoscope, or deeper suction through the bronchoscope for lower level respira-

*Supplied by the Lederle Laboratories Division of the American Cyanamide Company, Pearl River, New York.

tory tract obstruction. Laryngoscopy and bronchoscopy may have to be repeated several times and may obviate a tracheotomy, but such procedures require trained personnel available at all times.

Nothing is given by mouth to any patient with pharyngeal paralysis until the fever, irritability and drowsiness disappear. Maintenance of body fluids and physiologic balance can be carried out by parenteral administration of saline, glucose and nutrients, such as casein hydrolysates, preferably by the intravenous route. One must caution against the administration of too much fluid by vein because of the danger of over hydration and the likely production or increase of cerebral edema. Slight dehydration is preferable to over hydration. The experience of some clinicians favors the use of hypertonic solutions, such as glucose, to combat the edema which may be present in the brain and cord.[27] We have used 10 per cent glucose intravenously, but it is difficult to evaluate its effect.

Nasal gavage feeding may be instituted cautiously when the patient is afebrile, and there is no extension of paralysis, usually 4-7 days after admission. Within a few days thereafter, when the patient is able to swallow his saliva, he can be given small amounts of liquid nourishment by mouth starting with sips of water, and then tea, dilute fruit juices or clear broth. If these are well tolerated, soft foods may be tried.

Drugs, such as atropine, which have been used to dry up secretions, are harmful, they make secretions more viscid and more difficult to remove. Sedative drugs, such as the narcotics and the barbiturates, should not be used since they mask the symptoms of progression of the disease and in themselves produce respiratory depression. However, occasionally in the extremely restless and apprehensive patient in whom oxygen deficiency has been ruled out or corrected, the careful use of sedatives may be helpful. Stimulant drugs are often indicated in severe respiratory or circulatory depression—caffeine to stimulate the respiratory center, and epinephrine, or preferably neosynephrine for the falling blood pressure and failure of the circulatory center.[27] In some hospitals penicillin is given regularly to all bulbar patients with severe involvement to prevent infections of the respiratory tract.

Oxygen is given to every patient with severe paralysis of the palate or the pharynx even though there may be no cyanosis. It

is usually administered by nasal catheter which when properly inserted does not irritate the pharyngeal mucosa nor cause increased secretions. One must be careful to note that the tube does not block off. Oxygen by tent or face mask is not desirable—the patients object to the confinement and there is interference with proper observation of the patient and the necessary suction of secretions from the oropharynx. Oxygen and helium mixtures may be used for short periods.[38]

Two aids in oxygen therapy are the oximeter and the tracheotomy inhalator. The oximeter consists of a small device containing a photoelectric cell which can be attached to the ear, and a galvanometer which registers variations in arterial oxygen saturation. It provides a bedside method for determining the effectiveness of oxygen therapy without the need of blood samples and gas analyses, and it permits the recognition of hypoxia more accurately than clinical observation.[26, 27, 28] The tracheotomy inhalator provides for the administration of oxygen properly humidified, and under positive pressure if it is so desired. Suction, when necessary, can be applied through the small four-way tube which is attached to the tracheotomy tube.[29]

In those cases where there is spinal involvement too, bladder and bowel dysfunction require careful attention. Retention of urine requires catheterization, and drugs, such as prostigmine and furmethide,[40] may stimulate voiding. An indwelling catheter is rarely necessary. Sulfonamide drugs, but preferably the antibiotics, may be used to prevent or treat urinary tract infection. Enemata will aid bowel evacuation; cathartics are usually not effective. Gastric dilatation may occur especially in respirator patients. This can be serious aside from adding further insult to the breathing difficulty. Passage of a stomach tube and suction of the excess air can be of great advantage.

Reassurance of the patient, who is disturbed by the effects of the disease, or by an awareness of the seriousness of the illness, is most desirable. A comfortable and cooperative patient is important for effectiveness of treatment, and more radical procedures may therefore be obviated. Rest and sleep are important, and while no essential treatment should be omitted, there should be a minimum of handling of the patient.

Tracheotomy. The principle of tracheotomy to relieve some of

the symptoms of bulbar poliomyelitis and to prevent complications is not new,[11] even though few reports appeared in the literature until relatively recently.[2, 4, 14, 15] The value of the operation lies in the fact that tracheotomy by-passes secretion, it permits more thorough suction of the trachea and more effective oxygenation through the tracheotomy tube.

There is no general agreement as to the indications for tracheotomy in bulbar poliomyelitis, although all workers feel that it is a necessary emergency procedure in the rare condition of bilateral abductor paralysis of the vocal cords where there is mechanical obstruction to the interchange of air. However, there seems to be some agreement that the procedure warrants consideration in certain situations where it may be difficult to maintain an adequate airway by postural drainage and suction. These situations may arise in the following patients: 1. The very drowsy or stuporous patient who disregards his secretions. In this patient there is the constant threat of aspiration of secretions with resultant attacks of choking. 2. The excited patient who is uncooperative and resists suction and in whom fatigue is becoming a serious problem. In both these patients (1 and 2) progressive hypoxia probably contributes to the encephalitic symptoms. 3. The bulbar patient, with difficulty in breathing either from central or peripheral paralysis who must be given relief in the respirator. In this patient maintaining an open airway is especially difficult. There is also the danger of secretions being sucked into the trachea and lower respiratory tract by the action of the respirator, or the development of pulmonary edema from forced breathing against obstruction in the airway.

The lack of trained personnel to properly suction and keep the airway clear has been suggested as an indication for tracheotomy. This could arise during epidemics when the admission rate is high.

The question of tracheotomy and especially its indications and the frequency with which it should be resorted to is still open to debate. Some say that it is indicated only in rare instances; that it is a procedure accompanied by serious complications; and that it adds to the nursing and medical problems already present. Others say that only by doing many elective or prophylactic tracheotomies before the patients are in extremis can mortality be lowered; that careful observation of the patient is most desirable

but that there is a tendency to wait and resort to the procedure when it is too late to be of benefit; that if done early and therefore more carefully complications, such as pneumothorax, mediastinal emphysema, and difficult decannulation which is often due to incision through the larynx or the first ring, are minimized; and that the danger of infection can be controlled by antibiotics.

The operation is usually performed under local anesthesia preferably with a bronchoscope, or endotracheal tube in place. The tracheotomy tube should be inserted through the 2nd or 3rd rings. If a respirator is necessary, there are machines which permit care of such patients; the portable collarless respirator allows more room for action.

TABLE 3. *Tracheotomy in Bulbar Poliomyelitis.*

Locality	Year	Bulbar Cases	Tracheotomy		
			Cases	Deaths	Case Fatality Rate Per Cent
Willard Parker Hospital, New York City	1947	8	2	2	100.0
	1948	45	5	3*	60.0
	1949	126	6	6	100.0
University of Minnesota Hospitals[2]	1946	400**	75	46	60.1
Los Angeles County Hospital[5, 18]	1946	129	14	7	50.0
	1947	...	8	4	50.0
	1948	248	"over 60%"	..	...
	1946-1948	783	233***	..	...

*1 of these died 54 days after tracheotomy.
**"Approximately."
***"In 284 cases with obstruction to a free airway."

At the Willard Parker Hospital (Table 3), in 1947, 2 of the 8 bulbar poliomyelitis patients were tracheotomized and both died. In 1948, 45 patients had bulbar involvement and tracheotomies were performed on 5 severely ill patients. Two of these died within a short period after the operation, and a third, a bulbospinal respirator case, died 54 days later. Two patients survived. One, a 26-year-old woman with the rare bilateral abductor paralysis of the vocal cords, was well at a recent follow-up examination except for mild sequelae of bulbar poliomyelitis. The other, an 18-year-old girl with severe bulbospinal involvement, required prolonged

respirator treatment and died 13 months after the tracheotomy. Although it is difficult to judge, it would seem that three patients were definitely aided by the tracheotomy, and very likely the procedure helped to carry them through their acute illness. It is felt that the operation did no harm in any instance.

In 1949, 126 patients had bulbar involvement, and six tracheotomies were performed. None of these six patients survived. In two instances the operation was performed relatively early although the patients were very ill, with apparent relief following the procedure. All the bulbar patients were carefully observed and we attempted to select those who might benefit by a tracheotomy. Possibly the operation could have been done on others who went on to die, but on the other hand, many very ill patients whom we thought might have the operation did well without it.

Other institutions report on much larger series of cases (Table 3).

Respirator in Bulbar Poliomyelitis. The respirator is not often effective where there is bulbar involvement, although with severe central respiratory depression it may be of assistance. In the latter situation, phrenic nerve stimulation[43] may be a more effective method of producing artificial respiration than the conventional "iron lung." When there is at the same time involvement of the chest muscles or diaphragm or both in the bulbar patient the respirator is more clearly indicated. In such cases special attention must be given to keep the airway clear of obstruction, and frequent change of body position is important to prevent stasis. These patients usually are unable to cough and they have difficulty in raising their tracheobronchial secretions. Infection is ever a threat and complications, such as atelectasis, tend to occur. Tracheotomy may be indicated in some instances.

Portable chest respirators are of value for short periods and for emergency treatment. Other apparatus utilizing intermittent positive pressure breathing in conjunction with the standard respirators can be helpful to combat pulmonary edema. The rocking bed is a valuable apparatus for providing artificial respiration, and especially an excellent aid for weaning patients from the respirator.[43, 44]

Sequelae of Bulbar Poliomyelitis. That bulbar poliomyelitis has a better prognosis as regards ultimate recovery of function and

recovery from paralysis than the spinal type has been the generally held opinion. But a recent report[45] of patients followed up to 18 months after the acute illness indicates that a fairly large number had definite subjective complaints, such as nasal regurgitation, gagging on solid foods, and evident impairment of motor function of the palatal, laryngeal or facial muscles. The most frequent manifestation was nasal regurgitation, and some had persistent voice changes. The rate of recovery of function was similar to that of limbs affected by poliomyelitis, showing progress during the first six to 9 months and thereafter showing little or no improvement. Our experience at the Willard Parker Hospital in the past,[7] and our more recent experience, indicate that the sequelae of bulbar poliomyelitis do occur more frequently and are of greater significance than was formerly thought to be the case.

Tonsillectomy and Adenoidectomy and Poliomyelitis. Some reports from otolaryngological groups state that there is no causal relationship between poliomyelitis and tonsillectomy and adenoidectomy, such cases being coincidental. However, evidence seems to indicate that the incidence of poliomyelitis in recently tonsillectomized children within 30 days (even up to 60 days) following the operation is greater and that the bulbar type is usually the form of poliomyelitis to occur. This can happen only when the virus is either already present in the throat at the time of operation or introduced shortly afterwards. It seems advisable to defer elective operation on the nose, mouth and throat regions when poliomyelitis incidence is high, or is increasing.[46] This year we have encountered one patient, a boy of 5½ years, who developed symptoms of bulbar poliomyelitis 10 days after a tonsillectomy and adenoidectomy and died of progressive bulbar poliomyelitis four days after admission.

Transportation of the Acute Poliomyelitis Patient. Recent reports indicate that there may be some connection between physical activity and the extent of paralyses in acute poliomyelitis.[47, 48, 49] It has also been the feeling that the transportation of acute poliomyelitis patients over long distances may be hazardous, although in some cases it is unavoidable. This year at the Willard Parker Hospital five patients transported from camp or resort areas located as far distant as 100 miles died in relatively short periods after admission. One was dead on arrival, the others died in from

45 minutes to 12 hours after admission. All were bulbar poliomyelitis patients in critical condition on arrival, and all were transported by ambulance or private car. Their histories showed that they were not as severely ill when they set forth on their journey to our hospital as their condition on arrival indicated. There have been other similar instances in the past.

Parents may urge that the patients be sent back to their home city, but the hospital authorities in those areas, aware of the hazards of moving very ill poliomyelitis patients, should attempt if possible to overrule such requests if adequate facilities for treatment are available. In any case, if such patients are transported they should be accompanied by attendants and equipment sufficient to handle any emergency that may arise in bulbar poliomyelitis.

Poliomyelitis "Teamwork." I should like to emphasize that the treatment of the poliomyelitis patient, and especially the bulbar patient, is not a one person problem. From the beginning the internist or pediatrician, the nurse, and the physical therapist must combine all their skills to ensure the best possible care for the patient. Cooperation of the other specialists, such as the otolaryngologist, the orthopedist, the neurologist, the psychiatrist and the psychologist is also necessary. The otolaryngologist has an important role, especially when clearance of the air passages becomes a problem and the question of tracheotomy arises. His experience should be sought not for the surgical procedure alone but as a part of the team in the early treatment of the patient.

Not only the physical needs of the patient require attention as treatment progresses, but the psychological, occupational, recreational and educational needs require consideration. A happy, alert, cooperative patient responds best to the treatment of his disabilities. Nor must one overlook the parents and the family, for a proper attitude on their part reflects itself in the patient and in his response to rehabilitation.

SUMMARY

1. The clinical aspects of bulbar poliomyelitis are reviewed with emphasis on present day concepts.

2. Bulbar poliomyelitis accounts for almost the entire mortality attributable to poliomyelitis, but there are certain methods of treatment which will reduce mortality. These methods are discussed.

3. Thus far there are no drugs which will alter the course of the disease. Our experience with Darvisul (phenosulfazole) is discussed.

4. The controversial subject of tracheotomy in bulbar poliomyelitis is discussed.

5. Transportation of the acute bulbar poliomyelitis patient over long distances may increase the mortality rate.

6. Effectiveness of treatment of the bulbar poliomyelitis patient requires the cooperation of doctors and workers from allied fields, who form a "poliomyelitis team."

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CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York and Doctor Philip Moen Stimson, Cornell Medical School, New York. Other interested medical schools are cordially invited to submit student papers for consideration.*

NEWER CONCEPTS AND DEVELOPMENTS CONCERNING POISON IVY AND ALLIED CONDITIONS*

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Of the various types of dermatitides encountered in children, those of the contact variety are extremely prevalent. During the warmer months of the year, contact dermatitis due to exposure to poison ivy, poison oak and poison sumac is very commonly seen. Although the chemical nature of the toxicants found in these plants has been known for many years, actual knowledge concerning the fate of these toxicants, once contact with the skin has been accomplished, and knowledge of the defense mechanisms of the body, mobilized against these materials, have been extremely vague. As a result, no entirely satisfactory therapeutic regime has been devised and the only completely positive preventive measure is to avoid contact with the offending plants or their products. In the case of children, especially, this course is certainly not feasible. Therefore, the proper approach to the problem lies in an integration and correlation of known data, many of which are found widely scattered throughout various sections of the literature. With such information at hand, a more thorough understanding of the problem may be obtained, which may, in turn, lead to more effective therapeutic measures.

In addition to dermatitis venenata caused by the Rhus family of plants, resins and derived products from such members of the Anacardiaceae family as cashew nut shell fluid have been prepared in recent years.[1] These have caused outbreaks of contact dermatitis similar to that resulting from exposure to poison ivy, when objects made from or treated with these materials

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have been used. Wasserman and Dawson[2] have described 12 such toxicants in addition to poison ivy, some classified as Anacardiaceae and others from related species. Most of these substances originate from Oriental plants and their products as has been pointed out by Merrill.[3] Keil, Wasserman and Dawson[4] reported a case of dermatitis in a 13-year-old Puerto Rican girl, which developed periorally after the ingestion of mango fruit. This patient also demonstrated hypersensitiveness to poison ivy upon patch testing. Previously, Simmons and Bolin[5] had demonstrated that the stem sap, which many times may contaminate the peel of the mango fruit, was the source of the toxicant, the fruit per se being innocuous.

In all these cases, the toxicants have been shown to be chemically closely related to urushiol, the toxicant of poison ivy, poison oak and poison sumac. All these substances are either mono- or di-phenolic materials having a long, unsaturated, straight side chain in the meta position to one of the hydroxyl groups. Evidently, the vesicant action of the molecule depends upon the fat-like property of the long side chain to penetrate the epidermis, and upon the catechol nucleus for the vesicant activity. This has been proven by the use of various synthetic substituted catechols or derivatives of the natural vesicants of the Anacardiaceae, Rhus and related families. In all cases, cross-sensitivity among these materials and poison ivy existed.

It has long been appreciated that, upon exposure to air, the saps of these various plants darkened with a proportional loss of toxicity. This was soon ascertained to be due to the atmospheric oxidation of the phenolic nucleus to colored quinoid substances which per se were innocuous. This has led to the topical use of oxidizing agents as one of the methods of approach in the methods of treatment of poison ivy and related dermatitides. Among the agents used in this manner have been ferric chloride, potassium permanganate and sodium perborate. These methods, however, have resulted only in partial success, and the reports in the literature have been controversial. For example, Witherspoon[6] reported excellent results with sodium perborate in a series of 70 cases. In acute cases he applied first iced sodium perborate compresses containing 1 per cent of the chemical. In average cases, he found that a greaseless ointment containing 10

per cent sodium perborate and 1 per cent phenol was very satisfactory. The sodium perborate oxidized the urushiol while the phenol served as an antipruritic. On the other hand, Shelmire[7] reported very poor results with 10 per cent perborate ointment.

Recently, Sizer and Prokesch[8] have studied the effect of the enzyme tyrosinase upon poison ivy oleoresins and extracts, on urushiol itself as well as upon related natural and synthetic toxicants. In this and in additional investigations,[9] these workers have shown that this enzyme can inactivate the toxicants both in vitro and upon the skin surface of animals and patients. This was accomplished by the actual oxidation of the offending substances to nontoxic quinones. As this enzyme is completely harmless to the skin surface as compared with other oxidizing agents in common use, hope has been expressed[10] that the use of this substance may find practical therapeutic application in the near future.

Another method of attack in the therapy of Rhus dermatitis has been through the use of symptomatic treatment with such agents as calamine lotion, or by using antihistaminics and antipruritics (benadryl, pyribenzamine, etc.). Blumenthal and Rosenberg[11] studied the effects of benadryl in 14 cases of poison ivy. They found that 12 of the 14 patients experienced marked symptomatic relief, and in the other 2 the relief was only moderate. In 3 patients, the lesions ceased spreading shortly after the initiation of benadryl therapy, and within 72 hours were cleared entirely. In the remaining 11 patients, the actual course of the dermatitis was not altered, despite the symptomatic relief obtained. On the other hand, Tweedall and O'Connor[12] found that pyribenzamine was of no value in preventing the onset of Rhus dermatitis in a sensitized individual, once exposure had occurred. They noted that this drug was only moderately effective in relieving the subjective symptoms of itching and burning. In another approach to the problem, Klasson[13] treated poison oak patients with ascorbic acid. He postulated that this substance counteracted the toxic actions of histamine and like substances which were released as a result of the action of the poison oak toxicant on the body. The best route of administration was found to be the intramuscular one, although in some instances, oral administration of ascorbic acid in tablet form acted in support of the

injection. The drug manifested its action first by a reduction in itching, this antipruritic effect making its appearance approximately 2 to 4 hours after the injection. In this case, a maintenance dose was given since the vitamin is rapidly eliminated from the body. The symptoms of the already contracted disease were relieved by this regime, the best results being obtained with those patients who presented edema, as of the eyelids and the genitalia. Another method of treatment involved the use of cryotherapy. Robinson[14] studied 101 cases of dermatitis venenata. He treated one with liquid air, four with solid carbon dioxide and 96 with ethyl chloride spray. Only 5 cases in this series failed to respond. In general, he found that recovery occurred within 5 days with cryotherapy. Ethyl chloride seemed the easiest to use. Along similar lines, Park[15] used steaming hot water (130° F.). This produced a mild first-degree burn concurrent with symptomatic relief.

After noting the protective effects of chloroamide (a chlorine derivative used in chemical warfare experimentation to prevent certain war gases and volatile substances from reaching the skin), Sulzberger, Baer and Kanof[16] suggested the use of this chemical in an ointment perparation as a prophylactic measure in the production of experimental poison ivy dermatitis. Prophylactically, however, the use of this material would present many practical difficulties.

The last general approach to the therapy of poison ivy and related dermatitides lies in the immunological aspects of treatment. For many years it was observed, especially by the laity, that many individuals seemed to have a natural immunity to the disease. Others claimed to have obtained immunity by the ingestion of poison ivy leaves or preparations thereof. From this has evolved the current practice of parenteral administration of Rhus extracts, and the use, to a lesser extent, of oral preparations to prevent the onset of poison ivy, once exposure has occurred, and to alleviate the symptoms once they have appeared. Unfortunately, neither of these prophylactic measures have proven uniformly effective, and in many cases, once the symptoms have appeared, injection of the antigen further aggravates the picture. For this reason, Howell[17] feels that such treatment is contraindicated in the treatment of acute cases. To date, however, this method has been one of the most commonly used by most physicians.

The antigen used for prophylactic immunization consists usually in a solution of oleoresin of the plant, prepared by extraction of the leaves with suitable organic fat solvents. However, investigations have recently been directed toward the development of antigens which would be more effective on the one hand and less dangerous on the other. Stratton,[18] in his guinea pig studies, has used various poison oak antigens which were extracted from the original plant material with various solvents. One of the extracts was separated into fractions. One of these fractions was found to be more effective when administered orally, and another more efficient when given intramuscularly. These studies were extended to patients[19] with results similar to those obtained in animal experiments. Strauss and Spain[28] prepared an alum-precipitated pyridine-ivy complex which was suspended in saline solution. The activity obtained with this preparation was similar to that of an equivalent dilution of the alcoholic ivy extract. This preparation may also be used with poison oak, poison sumac, primrose, pollen oils, etc. These workers found that sensitive patients treated prophylactically with the alum-precipitated, pyridine-ivy extract were relieved by the second to the fifth injection. Although they obtained poor results in a group of 12 patients treated prophylactically, the preceding year with alcoholic plant extracts, prophylactic protection was obtained when this same group was treated with the alum-precipitated, pyridine-ivy extract. A group of 16 other sensitive individuals noted a season practically free from poison ivy dermatitis, following similar prophylactic injections with this material.

The purpose of the antigen-injection method of treatment is to bring about an active immunization—the formation of antibodies specific for the antigen, urushiol, by the host. Interestingly enough, until very recently, no one has been able to demonstrate the presence of antibodies specific for the toxicants, once they were introduced either on the body surface in an actual exposure or after injection treament. Firstly, sensitization to poison ivy follows a pattern similar to sensitization of the animal or human subject to other substances. The first contact of the individual with the toxicant acts to sensitize the body to the action of the drug. Usually no lesion develops after this first contact. Upon subsequent exposure to the toxicant, however,

dermatitic symptoms will develop, this after a suitable incubation period. Thus, as with anaphylaxis, the second and following exposures to the antigen, serve as "shocking" doses. In many cases, additional exposures to the toxicant cause further sensitization of the individual with increasing severity of symptoms. Rarely does any immunity develop from such exposure, the mechanism being one of allergy rather than of immunity per se. It must be stated further, however, that one exception to the rule does exist. Cases have been observed in which primary exposure to the causative toxicant lead to the appearance of symptoms. This phenomenon may be the results of prolonged contact of the antigen with the skin surface with little oxidation of the contactant by the atmosphere, the time interval being sufficient for the sensitizing dose to become the "shocking" dose.

McNair[21] has considered the pathogenesis and immunology of contact dermatitides in some detail. He suggests that poison ivy toxicant may enter via the sebaceous glands, sweat glands, hair follicles, and, of course, the skin surface proper. Before the poison can enter the epithelium, it may undergo a certain amount of oxidation and consequent loss of toxic properties. This loss of toxicity, however, is relatively slight, as the oxidation process proceeds slowly. In the light of recent information concerning the oxidases of the skin, this early observation of McNair is very interesting.

In passing through the stratum corneum, some of the toxicant remains localized here, the result of adsorption and absorption. Therefore, the thickness of this skin layer is a very important factor in the susceptibility of the individual and of various parts of the body to attack by the poison. Hence, poison ivy rarely, if ever, attacks the palms of the hands or the soles of the feet where the skin is thickest. It is a well-known fact that the most susceptible areas have proven to be those where the skin is thinnest. After passing through the stratum corneum, the toxicant passes either through or between the cells of the other layers of the skin, gaining access finally to the stratum corium.

Landsteiner and Chase,[22] working with guinea pigs, concerned themselves with the mechanism of spread of the poison ivy antigen after contact with the body. They were able to demonstrate that an intact lymph supply was necessary for sensitization of

the animal, once contact with the antigen had been brought about. Previously, Hudack and McMaster[23] showed that the lymphatics of the skin consist of a large plexus in the superficial layers of the corium which is horizontally continuous. For example, when dyes are injected in one spot, they spread through abundant anastamoses to distant areas. In addition, it is known that lymph serves as the main conveyor of fatty substances absorbed in the villi of the intestines. Therefore, it is not beyond the realm of possibility that urushiol, perhaps in an altered state as a haptene, or in the form of a complex with other substances such as glucuronic acid, sulphur-containing substances, phosphatases, etc., circulates through the body, after first entering the lymphatics of the skin. This supposition has been recently borne out by the experiments of Crepea and Cooke.[24] These workers sensitized guinea pigs to poison ivy by cutaneous application of the antigen. They were able to demonstrate the presence of antibody against poison ivy in saline extracts of washed splenic cells. In addition, successful passive transfer of dermatitic sensitivity was attained in 7 of 15 trials with serum, and 16 of 19 trials with a suspension of splenic cells.

From the above data it follows that the antigen in the course of its travels through the lymphatics finally gains access to the lymphoid tissue of the body. Chase, White and Dougherty[25,26] and Ehrich and his coworkers[27] have recently demonstrated the presence of antibody globulin in the lymphocytes of lymphoid tissue. Dougherty, Chase and White[28] have shown that dissolution of lymphocytes within lymphoid tissue by the administration of adrenal cortical extract or by the physical extraction of lymph nodes has released a protein identical electrophoretically with serum gamma globulin. In view of these data, it may be that exposure to poison ivy or related toxicants may result in the production or at least the storage within the lymphoid tissue of antibodies specific for the antigen in question. Therefore, the immunization methods so widely utilized in poison ivy therapy may have their bases in the foregoing principles, although the practice, in this case, preceded the actual demonstration of the mechanisms.

In contact dermatitides, however, the above is not believed to be the only means of body defense against the poison ivy antigen

As has been mentioned, oxidation of the urushiol in the skin to nontoxic substances is believed to occur to some degree. Toyama,[29] in 1918, stated that the urushiol was oxidized to a certain degree by the oxidases, such as laccase, which are present in the plant material itself; and also by the oxidases normally present in the skin. Keil, Wasserman and Dawson[30] believe that the hydroxyl groups of the urushiol are oxidized in the skin to a quinone configuration or are somehow combined with native protein in the manner propounded by Landsteiner in his theory of haptenes. McNair[31] further suggests that the oxidases of leukocytes and detoxifying action of the iron in hemoglobin contained in erythrocytes may play a role in this action.

The above considerations lead to the question of the nature of the oxidases of the skin, the latter being the first barrier that the antigen has contact with upon exposure to the body surfaces. Recent work has demonstrated the presence of tyrosinase and similar polyphenolases in the skin. It is well-known that the melanoblasts of the epidermis of higher animals contain an enzyme which some authors describe as tyrosinase, while others refer to it as "dopa oxidase," which oxidizes 3, 4-dihydroxyphenylalanine ("dopa") to the darkly pigmented substance, melanin. Whether melanin is one substance or a mixture of quinoid substances is not known. Whatever the nature of the oxidases of the skin, the fact remains that they are present and are potentially capable of acting upon phenolic substances similar chemically to urushiol and related toxicants.

In a very significant paper, Ginsburg[31] was able to demonstrate the presence of a polyphenol oxidase in the skins of guinea pigs. He found this enzyme in the globulin fraction of the skin, and was able in turn to concentrate it from crude extracts. In light of the aforementioned data concerning the relationship of lymphocytes and lymphoid tissue and antibody globulin, this is of special interest. Ginsburg presented data demonstrating that boiling destroyed the activity of this oxidase. From his studies, he concluded that the enzyme in question was either tyrosinase or any enzyme very similar to it. Further, the same author was able to demonstrate an inhibitor which completely prevented the action of the polyphenol oxidase present in the skin. The relationship of enzyme to inhibitor was shown to be controlled genetically;

albino guinea pigs have a larger quantity of inhibitor than enzyme.

The above findings seem to be in agreement with earlier observations of Gortner,[32] the latter defining the oxidase as "tyrosinase." This author also emphasized the genetic influences responsible for either oxidation of the chromogen by the enzyme to a pigment, or partial or complete inhibition of the enzymatic action to form various hued pigments varying from a dark color to pure white. In the latter case the enzyme was completely inhibited.

The foregoing discussion is very pertinent in light of the observations of Chase,[33] who has recorded that the susceptibility to skin sensitization to certain chemical compounds in guinea pigs was under genetic control. He was able to breed a strain of guinea pig which was sensitive to a simple compound, 2, 4-dinitrochlorobenzene, while another inbred strain was not affected by this substance.

Recent work has substantiated the above data. Flesch and Rothman[34], in a preliminary report, studied the oxidase-inhibitor system in the skin. They demonstrated that the inhibitor substances, which were apparently identical to the ones described by Ginsburg, were sulfhydryl compounds, and that these were inhibitory in their action to the formation of melanin-like pigments in the skin. In further studies, Flesch[35] demonstrated that this inhibitor was counteracted by the action of cupric ions. Since copper is known to be the inorganic activator of the enzyme, tyrosianase, this author used the concentration of cupric ions as a measure of tyrosinase or oxidase activity of the skin; and he used the concentration of sulfhydryl groups as a measure of inhibitor action. Thus, in a study of the role of copper in mammalian pigmentation, Flesch[36] showed that the epidermis had a much higher copper content than did the corium. This observation may have its explanation in the fact that the melanoblasts are found in the epidermis. Melanin obtained from mouse malanomata was also shown to be relatively rich in copper content. This author also states that, in general, black and gray hairs of rabbits, rats and guinea pigs usually contained more copper than was found in the white hair of the same animal.

It may be concluded from the data presented that the individual inherits genetically the ability to manufacture a particular amount of enzyme in relation to an enzyme-inhibitor. The ratio of enzyme to enzyme-inhibitor varies from individual to individual, and is

characteristic of the species in question. One may, therefore, classify guinea pigs and other laboratory animals into gradations of color ranging from white to black, and each hue is the result of a characteristic ratio of the phenolase to the inhibitor.

It may be postulated that those individuals who inherit greater quantities of the polyphenolase in relation to the specific inhibitor in their skins and cells may possibly be more resistant to the permeation of the toxic material responsible for Rhus dermatitis, since the antigen is detoxified in the skin through the process of oxidation. This may then account for the relative immunity of some individuals to Rhus dermatitis as compared to others who show definite susceptibility.

The fact that oxidation destroys the phenolic compounds of Rhus oleoresin has been the basis of the use of such topical oxidizing agents as ferric chloride, potassium permanganate and sodium perborate, described previously. The therapeutic end results obtained through the use of such materials may have a mechanism similar to the action of the body polyphenolases on the poison ivy toxicant within the skin or the body itself. Methods utilizing the parenteral administration of actual Rhus antigen dispersed either in a solvent or in a bland oil, in view of the previous discussion, may act to produce antibodies against the toxicant, and, at the same time, may cause a change in proportion of skin oxidase to inhibitor, bringing about a decrease in the latter substance. The result is the production of immunity to Rhus dermatitis.

A discussion of the question of the relative resistance of individuals with higher skin oxidase content to Rhus dermatitis as compared with those with lesser amounts of the enzyme follows. In general, at the present time, the data available are not too accurate, and may be confusing. McNair[21] compared the susceptibility of blondes and brunettes to poison ivy. His impression is that blondes are more susceptible, although the results of other workers are controversial. Deibert[37] et al., on the other hand, concluded that skin color was not a factor in poison ivy susceptibility. This author reached this conclusion after comparative investigations of poison ivy susceptibility in full-blooded American Indians and white subjects. The patch test was used as a criterion of susceptibility. This is in contrast to the findings of Lain[38] who noted that the skin of the full-blooded American Indian is practically

immune to poison ivy and other plant dermatitides. In a similar study, Coca, Deibert and Menger[39] questioned 11 physicians and one superintendent of an Indian school. The findings recorded confirmed those of Lain. However, in the latter study, the authors noted that full-blooded American Indians are much less subject to allergic phenomena in general than is the white population.

Cocheu, Boyd and Speer[40], in a survey of the incidence of contact dermatitis in an industrial plant, found a preponderance of incidents among the blond employees as compared with the brunette. The question of skin color as a factor in poison ivy susceptibility can be answered only after carefully controlled surveys have been made, and evaluation of the data obtained therefrom has been carried out.

In considering the immunological aspects of Rhus dermatitis, no discussion would be complete without reference to the reaction of the hypersensitive individual to the antigens. Until recently, little attention has been given to the possible dangers or sequellae to Rhus dermatitis. However, Rytand, Burnham and Cox[41] reported a case of probable periarteritis nodosa which followed an attack of poison oak. Rytand[42] has also noted the appearance of renal disturbances in a series of 7 patients. The symptoms were noted either during an attack of poison oak dermatitis or shortly after recovery. One of the cases resembled lower nephron nephrosis syndrome and terminated in fatal anuria; 5 other cases showed a predominance of renal tubular degeneration. Of these 5 cases, 4 showed the nephrotic syndrome while 3 showed pure lipoid nephrosis. The remaining patient in the series demonstrated primary stages of glomerular nephritis. Since there was a complication—a previously incurred streptococcal infection—no conclusions could be drawn in this particular case. Significantly, Rytand suggests that the poison oak toxicants may bring about the antigenicity of renal tissue with the resultant production of auto-antibodies.

By and large, the sequelae of Rhus dermatitis described above may be included in a vast list of diseases tabulated by Selye[43] under the heading of the general adaptation syndrome and diseases of adaptation. This worker claims that many "shocking" stimuli are capable of bringing about conditions similar to if not identical with periarteritis nodosa. Further, Selye describes the development of renal pathology after exposure to stress or trauma, acting through

the median of the adrenal cortex. That Rhus toxicants or body conjugates or their products may act as shocking agents in certain hypersensitive individuals does not seem unreasonable; therefore, they may account for the more serious sequelae observed occasionally, and perhaps, the edema and other generalized symptoms. On the other hand, the generalized symptoms may be due to the toxicants per se or a combination of all these factors.

In conclusion, it may be stated that Rhus dermatitis fits into a broader general picture in which many immunological, allergic and biochemical factors play an important role. More successful methods of prevention and treatment may follow a more thorough understanding of these factors.

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PRIMARY ATYPICAL VIRUS PNEUMONIA IN CHILDREN. (Maandschrift voor Kindergeneeskunde, Leyden, 17: 172, 1949). Keizer discusses the clinical aspects and complications of virus pneumonia on the basis of observations on 41 children. The insidious onset is noteworthy. Roentgenographic examination is of diagnostic value. The cold agglutination is not specific but is of help after hepatic and other diseases have been excluded. Repeated determination of the cold agglutination titer is of diagnostic and prognostic value. Although the mortality rate is zero, virus pneumonia is fairly serious in children because of such complications as hemolytic anemia, hepatitis, acute hemorhagic nephritis, myocarditis, endocarditis, encephalitis, benign reticulosis, acute suprarenal insufficiency, mastoiditis, sinusitis and the exanthems. Sulfonamides and penicillin counteract secondary infections only, and their use involves the risk of anemia. Gamma globulin in doses of 0.2 cc. per kilogram of body weight helped in some cases but did not always prevent relapse and complications.—*J.A.M.A.*

THE "STOMACH" AND "EYE" TEETH

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The younger men in pediatrics may wonder why the lay public still refer to the deciduous canine teeth as the "stomach" and "eye" teeth.

Infectious and putrefactive diarrheas of the past were known as summer complaints. Diarrhea of second summer, cholera morbis, cholera infantum, etc., were seen not too many years ago with a frightening mortality.

Women who had adequate breast milk would not wean the infant during the late spring or summer for fear of the hot weather plus the unknown factor of infected raw bulk milk causing a fatal diarrhea. Many infants were kept on the breast until the second fall of life, which meant 12 to 18 months. During this time they received practically nothing except breast milk which was an obviously deficient diet. In its favor was the fact that a greater percentage of them lived, in comparison to the weaned or entirely bottle fed infants.

The diarrhea so prevalent 30 years ago would occur at any time during the year but the largest number occurred during the summer, particularly when the heat was intense. Manure, overripe fruit, fish markets in the street, were responsible for the presence of innumerable flies to contaminate the milk. It was coincidental that this condition occurred at the time when the child was trying to erupt the lower canine teeth. Some of us have felt that cutting teeth in some children is accompanied by looseness of the bowels with definite relief from lancing the gums. The severe diarrheas of the past were associated by the lay public with the lower canines which were called the "stomach teeth."

We find most infants cutting the lower canines first. It is usually a few days to several months later that the upper canines erupt. In a severe, protracted case of infectious diarrhea in the past, those infants who survived, the absorption of toxins was so great that by the time the child was getting well his eyes would suddenly "turn in." This bilateral (usually) strabismus occurred about the time the upper canines were being cut hence the lay term "the eye teeth." The teeth obviously had nothing to do with the eye but the toxin producing the third nerve paralysis was to be blamed.

There is no other explanation that I know of for the lay public calling the deciduous canine teeth the "stomach" and the "eye" teeth.

15 *E. Arrellaga Street.*

ETHER TEST IN DIAGNOSIS OF VENOUS-ARTERIAL SHUNT. (Archives des Maladies du Coeur, Paris, 42: 601, June 1949). Donzelot and co-workers performed the ether test of Hitzig and and Benenson on 500 patients between the ages of 1 and 45 years with congenital heart disease. Three hundred and fifteen of these patients (63 per cent) had tetralogy of Fallot. The amount of ether injected into the antecubital vein or into the external juglar vein in small children varied from 0.03 cc. to 0.5 cc. The reaction to the test was positive in 84 per cent of the patients tested and negative in 16 per cent. Seventy-five per cent of the tests which elicited positive reactions were performed on patients with tetralogy of Fallot. A positive reaction to the ether test is pathognomonic of a venous-arterial shunt, or at least of a mixed shunt. A negative reaction to the test presents reliable proof of the absence of the shunt. The existence of the shunt was verified in 27 instances by necropsy. Angiographic examination was performed in 102 instances, and it confirmed the existence of the shunt in 75 instances in which the reaction to the ether test was positive and the absence of the shunt in 27 instances in which the reaction was negative. The results of the ether test were confirmed by surgical intervention in 129 instances. The ether test is of considerable value in the diagnosis of congenital heart disease in patients without cyanosis or with doubtful or late cyanosis. It also is of value in the diagnosis of Eisenmenger's syndrome and in the differential diagnosis of either Roger's disease or interauricular communication. The test should be made systematically on all patients with congenital heart disease as an aid in the diagnosis of the arterio-venus shunt.—*Journal A.M.A.*

CLINICAL-PATHOLOGICAL CONFERENCE

WILLARD PARKER HOSPITAL, NEW YORK

Meeting held March 21, 1950

DR. PHILIP MOEN STIMSON, presiding.

PRODROMAL MEASLES. INTERSTITIAL PNEUMONIA.

DR. ELSNER presented Case No. 583, an 8-month-old colored male, who was admitted to Willard Parker Hospital at 9:25 A. M. on February 4, 1950. For three days he had had fever and cough. On February 4, 1950, a sibling developed a measles rash.

This patient was seen and referred by another hospital at which he was found to have wheezing respirations. A diagnosis of asthmatic bronchitis and exposure to measles was made.

Physical Examination on Admission here showed a fairly well-developed, thin, colored male with a temperature of 101.6° F. and respirations 28 per minute. His palpebral conjunctivae were injected. Both tympanic membranes were pink and there was cerumen in the canals. His tongue was coated and his pharynx injected. There were a few rhonchi scattered throughout both lungs which cleared with coughing. The cough was loose. There was no lymphadenopathy. The skin was hot and dry with no rash. No Koplik's spots were seen in the mouth, although the buccal mucosa was reddened.

Clinical Diagnosis was prodromal measles; mild bilateral otitis media, acute catarrhal.

Course. The patient was considered moderately sick. Vaccination, Schick and control, Mantoux, and 2 cc. gamma globulin were given immediately after admission.

On February 5, 1950, the patient became very sick at 2:00 P. M. His temperature was 105° F. He had several loose stools and appeared dehydrated. At 2:30 P. M. he was given 1 cc. of procaine penicillin in oil and 1 gram of sulfadiazine to be followed every 6 hours by 5 grams. At 10:15 P. M., a 1,500 cc. clysis of 5 per cent dextrose in normal saline was started with Hydase at the site of each clysis. His respirations were 48 per minute then.

At 11:00 P. M. the respirations had dropped to 14 per minute

and were irregular, and at 11:15 P. M. respirations ceased. Adrenalin intramuscularly and artificial respiration were of no use.

Discussion. No x-ray studies were made on this patient. No blood count. When admitted patient was considered very sick. Diarrhea started that morning. Described as very loose stools.

Dr. Dolgopol. Pathologically, nothing remarkable revealed grossly. The lungs showed a few darker depressed areas suggestive of atelectasis, but they were otherwise air-holding. The heart and liver showed no gross pathology. The spleen was small and congested. The renal cortex was pale. There was a small agonal intussusception in the ilium, without any evidence of peritoneal reaction. Brain did not show anything remarkable.

Microscopic Slides were projected by Dr. Dolgopol. The lungs showed the picture of capillary bronchitis and peribronchial (interstitial) pneumonitis. Most bronchi were filled with polymorphonuclears and showed desquamation of the epithelium. The bronchial walls were infiltrated with lymphocytes and polymorphonuclears. In many areas of both lungs there was a marked thickening of alveolar walls due to their infiltration with mononuclear cells, but there was no intra-alveolar exudate. A few giant cells of measles type were found in the spleen and mesenteric lymph nodes. The liver showed moderate lymphocytic infiltration. No giant cells were found in the appendix or tonsils. Brain did not show any encephalitis.

PERTUSSIS. PULMONARY TUBERCULOSIS

Dr. White presented Case No. 5042, a 6-month-old female infant, who was first admitted November 17, 1949, to this hospital at the age of three months with a history of exposure to pertussis. This infant was again re-admitted on December 20, 1949 with a four-day history of recurrence of paroxysms and vomiting.

Physical Examination on Admission revealed a temperature of 100° F., dullness and crepitant râles over the left base and scattered course râles over both lung fields. Typical paroxysm was observed. Penicillin was ordered since patient's temperature continued to rise. Streptomycin was added on December 25, 1949. X-ray on first admission, November 17, 1949, showed "a small patch of consolidation in each lung near the hilum." X-ray on December 21, 1949 showed little change from the previous x-ray.

Course. Child seemed comfortable and was gaining weight but

continued to run a low grade fever though physical examination revealed no abnormalities. Streptomycin was stopped on December 25, 1949.

Chest x-ray on January 9, 1950 showed "extensive consolidation of the right lung, involving chiefly the upper lobe." On January 12, 1950 aureomycin therapy was begun. Child continued to have a fever and x-ray of January 17, 1950 showed no improvement in the pulmonary lesion. Aureomycin stopped January 20, 1950. It was learned that a sibling of the child had tuberculosis and a patch test on January 28, 1950 was positive.

Bronchoscopy on February 6, 1950 showed copious mucoid secretion obscuring the main bronchi. Infant was asymptomatic immediately after the procedure. Several hours later her temperature went up and the next morning she showed respiratory difficulty with retraction and cyanosis. This did not respond satisfactorily to oxygen, steam or suction. On February 8, 1950 an airway was inserted and a tracheotomy performed. There was immediate improvement in the child's respirations.

The child continued to appear desperately ill with temperature spikes up to 108.2° F. and vomiting brownish material. Parenteral fluids, oxygen and streptomycin were given. Signs of progressive collapse of the left lung were noted on February 9, 1950 and at 9:30 A. M. on February 9, 1950 the child expired.

Dr. Dolgopol presented gross specimens. The greater part of the right upper lobe presented the picture of caseous pneumonia. Caseous lymph nodes were present in the right hilus. The greater part of the left lung and large portions of the right middle and lower lobes were dark red in color, non-airholding and apparently atelectatic. A few scattered miliary tubercles were present in both lungs, liver and spleen. There was no meningitis.

Microscopic Slides were shown. The caseous part of the right lung presented confluence of numerous tubercles. The alveolar walls were not seen any more. The walls of many small bronchi were involved in the process. Miliary tubercles were present in other parts of the lung. In the left lung, mucus and polymorphonuclears plugged the lumen of many bronchi and the lung tissue was partially collapsed. Rare tubercles were seen. Only right hilar nodes were caseous. Miliary tubercles in the spleen and liver were fairly numerous.

ACUTE RIGHT CERVICAL ADENITIS

Dr. Ross presented Case No. 736, a 3-year-old girl, who was admitted to this hospital at 3:00 P. M. on February 11, 1950. She had been seen earlier in the day at Mt. Sinai and referred with a diagnosis of measles and cervical adenitis.

Her brother had had measles two weeks previously. The child had been well until about four days before admission. During this period she had a cough, a large tender swelling of the right side of the neck appearing on the morning of the day of admission. Koplik's spots were reported to have been present at Mt. Sinai; no rash was seen there.

Physical Examination on Admission to Willard Parker revealed an acutely ill child. Temperature was 101.2° F. There was moderately severe conjunctivitis. Both ear drums were red. There was a "faint equivocal erythema over the face, trunk and arms." There was a question of whether or not Koplik's spots were present. The throat was acutely inflamed, the tonsils and adenoids large. Oral hygiene was very poor. Brawny swelling of the entire right cervical was present. No discrete masses or nodes were palpable there.

Clinical Diagnosis on Admission was acute right cervical adenitis, and possible measles; peritonsillar abscess, mastoiditis, Vincent's angina and Steven-Johnson syndrome to be ruled out.

Course. 300,000 units of procaine penicillin were given immediately. By 4:00 A. M. of the following morning, the child was in slight respiratory difficulty, which was felt to be due to mechanical interference by the local edema of the throat. Child was slightly hoarse then, so that steam was ordered. She was taking fluids well, orally. Her temperature was brought down a little from 106° F. but by 10:00 A. M. was back to 107° F. The child was using accessory muscles for respiration more than previously. The swelling of the right side of the neck was about the same. Later in the day, however, it became more extensive and spread to the left side and suprasternally. Examination in the morning showed no superficial abscess in the pharynx and no swelling of the pharyngeal wall or tonsils. The anterior surface of the epiglottis was bulbous and pale. It was felt that the child had a deep abscess, probably in the pharyngomaxillary space.

Large doses of aqueous penicillin, parenteral and oral sulfonamides and a clysis were given during the day.

Because of the persistent fever, increasing local swelling and marked increase in toxicity, a surgical exploration of the right cervical area was performed. Minimal nitrous oxide anesthesia was used. At operation, the tissues were soft and edematous, exuding serous fluid. No induration, cellulitis, or localized disease process was found. Exploration of the pharyngomaxillary and the carotid spaces was performed. Respirations postoperatively became slower and about one-half hour postoperatively, the child died.

Discussion. Dr. Dolgopol. At autopsy, no rash was seen on the skin. Autopsy was limited to "wound inspection." The surgical incision was 2½ inches long. We had to undermine the skin. I succeeded in removing the tonsils, larynx, cervical lymph nodes, small portions of the right parotid gland and small pieces from the upper lobe of the right lobe. There were numerous congested cervical lymph nodes.

Dr. Dolgopol presented the gross specimens. The tonsils were dark-red in color. The soft palate was less congested. On pressure, thick green pus appeared from the crypts. The larynx and trachea also were congested. There was no obstruction of passages.

Microscopic Slides were projected. The tonsils were congested. The epithelium was present and there was no membrane. Many capillaries were partly thrombosed. The crypts were filled with mucus. Polymorphonuclears were absent in the plugs or in the stroma of the tonsil, but some were present in blood vessels. A number of unusually large lymphocyte-like cells were seen in the stroma of the tonsil, between normal lymphoid follicles. Clusters of cocci and many polymorphonuclears were present in the tissue surrounding the cervical lymph nodes, in the area of operation, but the lymph nodes contained mostly large lymphoid cells. The picture of lymph nodes and tonsil resembled that of infectious mononucleosis, but in the absence of hematological and serological work on the patient, this could not be confirmed.

A staphylococcus aureus (hemolytic, coagulose positive) and alpha streptococcus were cultured from all tissues. Both were resistant to sulfanomides. The streptococcus was moderately sensitive to penicillin and streptomycin. The staphylococcus was resistant to penicillin and highly sensitive to streptomycin.

DEPARTMENT OF ABSTRACTS

COLEMAN, L. L.: THE PSYCHOLOGIC IMPLICATIONS OF TONSILLECTOMY. (New York State Journal of Medicine, 50:1225, May 15, 1950).

Tonsillectomy is one of the commonest procedures of childhood. Since this operation is usually an elective one, the child should be better prepared mentally and physically for the surgical assault. The immediate and delayed reaction may be one of many alterations in behavior, such as family antagonisms, negativisms, night terrors, hostilities, phobias, obsessions and other phychoneurotic patterns. One can minimize the psychological impact by better comprehension, education and planning. To obviate the psychological pitfalls associated with tonsillectomy, the author recommends the following: (1). The child must be given a calm, forthright, and honest explanation of the reason and need for the operation. (2). The child must be spared the tension and anxiety reflected by the attitude of the parents. (3). Avoid the element of surprise and confusion. (4). Explain that the anesthesia is to spare the child pain and that the forced sleep is of very short duration. (5). The presence of one or both parents during the immediate preoperative and postanesthetic period is essential. (6). The doctor who is familiar with the child should escort him to the operating room since the moment of separation of the child from the parents is one of the most traumatizing ones of the entire experience. (7). Permit the child to manifest overt hostility during the postoperative period. MICHAEL A. BRESCIA, M.D.

SARROUY, C. AND GILLOT, F.: THE TREATMENT OF TYPHOID FEVER IN CHILDREN WITH CHLOROMYCETIN. (L'Algérie Médicale, 54:98 Feb. 1950).

The authors treated nine cases of typhoid fever in children ranging in age from 14 months to 12½ years. There were eight recoveries and one death. The one death was in a boy of six years in whom treatment was started late and he succumbed in cardiovascular collapse. Of the eight cases that responded to the chloromycetin, four were afebrile 48 hours after beginning the treatment, the temperature subsiding by crisis. In the others, the tempera-

ture was normal by the fifth day. The amount of chloromycetin used varied from 4 to 20 grams per case depending upon the rapidity of response. MICHAEL A. BRESCIA, M.D.

KYLE, J. L.: OCULAR FINDINGS IN CONGENITAL HEART DISEASE. (Canadian Medical Association Journal, *62*:263, March 1950).

The author reports on the ocular findings of 30 cases of congenital heart disease. The cases included the tetralogy of Fallot (17 cases), patent ductus arteriosus (7 cases), Eisenmenger's syndrome (3 cases), interventricular septal defect (1 case) and one case of undiagnosed heart defect. The principal ocular manifestation is the fundus picture and is apparently directly proportional to the polycythemia. The ophthalmoscopic picture consists of engorged and tortuous veins, of arteries which resemble normal veins and of a generalized purplish hue of the entire fundus. When the engorgement becomes so marked as to change the color of the fundus, it is referred to as "cyanosis retinae". In this series of cases, anterior axial embryonic opacities were noted in four cases and persistence of the anterior portion of the hyaloid artery occurred in two. One patient had bilateral coronary cataracts and multiple blue dot opacities. Another infant had a unilateral total cataract associated with other gross ocular defects consisting of microcornea and microphthalmos, heterochromia iridis and esotropia. In none of these cases of congenital cataracts and congenital heart disease could a history be obtained of the mother having German measles during the first three months of pregnancy.
MICHAEL A. BRESCIA, M.D.

LANDAU, D. B.; GOODRICH, H. B.; FRANCKA, W. F. AND BURNS, F. R.: DEATH OF CESAREAN INFANTS. A THEORY AS TO ITS CAUSE AND A METHOD OF PREVENTION. (Journal of Pediatrics, 36:421, April 1950).

From their observations the authors feel that the immediate clamping and cutting of the umbilical cord deprives the newborn cesarean infant of a considerable amount of blood. It is estimated that more than 26 per cent, but less than 34 per cent, of the average total fetal blood is in the placenta and umbilical vessels at the end of fetal life. The clinical course of these infants is

similar to that of older patients in shock from loss of blood. If the blood deprivation is beyond a critical point, symptoms of secondary or delayed shocks may appear two to four hours later consisting of grunting shallow respirations, air hunger, cyanosis, costal retraction and terminal convulsions. To salvage the blood which might be retained in the placenta and umbilical vessels the authors have recommended the following technique: The assistant designated to care for the infant is scrubbed and stands to the left of the operator. After the infant is deliverd from the uterus this assisant holds the infant by the legs with the head down. The pharynx is aspirated by a rubber bulb syringe. The cord is neither clamped nor severed. The placenta is separated from the uterus and wrapped in a large Turkish towel and secured by clamps. The infant and the wrapped placenta connected by the intact umbilical cord are carried to an awaiting table or cart. The placenta is suspended from a standard by means of a clamp attached to the towel. The cord is not cut until its blood vessels have collapsed, usually in from six to ten minutes

MICHAEL A. BRESCIA, M.D.

BUCKLES, M. G.: SURGICAL TREATMENT OF BRONCHIECTASIS IN CHILDREN. (Journal of the American Medical Association, 143:344, May 27, 1950).

It is estimated that bronchiectasis represents 5 per cent of all diseases in childhood. The classic symptoms are cough, sputum and hemoptysis. The patients are subject to numerous infections of the upper respiratory tract and recurrent attacks of pneumonia. The disease is chronic and progressive. There is evidence that patients in whom bronchiectasis develops before the age of ten do not live beyond the age of 40. It is generally agreed that the definitive treatment is surgical, the treatment of choice being early pulmonary resection. The author reports on the surgical treatment of 25 children ranging in age from 2 to 12 years. All gave a definite history of chronic cough and pneumonia. In most instances the onset of trouble started with measles, pertussis or pneumonia. The surgical procedures done in these 25 cases consisted of unilateral lobectomy in 21 cases, pneumonectomy in 2 cases and bilateral pulmonary resection in 2 cases. There was no mortality and the ultimate results were excellent in 16 cases

and considered good in 7 cases. Two patients with bilateral involvement, although improved by surgery, still have cough and sputum production. MICHAEL A. BRESCIA, M.D.

WU, W. Y. AND CHU, L. C.: CONGENITAL STENOSIS OF JEJUNUM. (Chinese Medical Journal, 67:527, Oct. 1949).

Congenital stenosis of the jejunum is quite rare since none has been reported in Chinese literature and Ladd and Gross reported only 2 such cases in a total of 21 cases of intestinal stenosis. The case reported is that of a male 24 days of age with a history of vomiting since birth. The vomiting became progressively worse and more frequent. Appetite was poor and there was loss of weight. The stools were scanty up to a week prior to admission to the hospital. On admission to the hospital the condition of the child was good except for some dehydration. X-ray examination with barium meal revealed definite evidence of stenosis in the upper part of the jejunum. At operation a rounded mass about 1.5 cm. in diameter was found in the upper part of the jejunum about 14.0 cm. below the ligament of Treitz. The intestine proximal to the mass was very much dilated and the distal loop was collapsed. A side to side anastomosis was accomplished. The infant withstood the operation well and eventually made an uneventful recovery. MICHAEL A. BRESCIA, M.D.

CIRRHOSIS OF THE LIVER IN NURSLINGS. (Maandschrift voor Kindergeneeskunde, Leyden, 16: 465, 1949). Dekker-Jonker presents observations of 2 nurslings who were the first children of a healthy couple. Both children died, one after six weeks and the other after eighteen days of life. Both had glycosuria and enlargement of the liver. Necropsy revealed fatty degeneration and beginning necrosis in both. Syphilis had been ruled out. There was no antagonism in the Rh factor in the parents. The bile passages were not obstructed, and no other congenital defects were present. The author believes that the cirrhosis must have had an intrauterine onset. He directs attention to reports in which dietary deficiencies are suggested as probable causes of cirrhosis of the liver and suggests that in further pregnancies the mother of these children be given a diet with high protein and low fat content —*Journal A.M.A.*

BOOK REVIEWS

FROM THE HILLS. AN AUTOBIOGRAPHY OF A PEDIATRICIAN. By John Zahorsky, M.D. Cloth. Pp. 388. Price $4.00. St. Louis: C. V. Mosby Co., 1949.

Although one might not consider this autobiography a well planned book, technically, it nonetheless has "heart" and feeling. If the purpose of an autobiography is to reveal the thoughts and motivations of the author then the volume has succeeded admirably. From humble beginnings which Zahorsky relates with humility and some pride he became one of the most successful pediatricians in America. He became a pediatrician not because of a desire to become a "big time" specialist but because of a true and deep love for children. Zahorsky prefers to refer to himself as a practical physician but he was a keen observer of his patients as well as of nature to which he has returned in his last years as a hobby of retirement. One can only envy a mind which can find pleasure and contentment in the finer aspects of life which in his case are nature and poetry. His observation was keen enough for him to be the first to describe roseola infantum. Others have chosen to designate the disease as exanthem subitum, although I cannot see the advantage in name change. It would be most fitting and proper to refer to this condition as Zahorsky's disease. In the third part of the book which the author has designated as "The Decline" there are a series of essays that reveal the simple and penetrating thinking of the author and are very worth while reading. Some of these essays are named Psychology, Mother Love, Genetics, Random Reflections, etc. The idealism and the castigation of some of our brethren is revealed in the chapter discussing income: "Nothing is more shocking than the outbreak of money madness in a doctor. It is the rock upon which the profession splits into professional doctors, merchant doctors and quacks. Too many doctors have the itch for easy money and charge exorbitant fees for trifling examinations, but the majority, in which class I belong, are more concerned with the medical service than with the magnitude of the fee. A child coming to any physician with some simple self-limited ailment, diagnosed

at a glance, can be treated perfectly well by any doctor. Though I have the greater reputation I feel like a robber when I extort a double fee on this account." This book can be read with pleasure and with profit. MICHAEL A. BRESCIA, M.D.

STILLBIRTHS. By Ian Sutherland, M.A. Paper. Pp. 91. Price $1.50. New York: Oxford University Press, 1950.

This is a statistical study of stillbirths in Great Britain and Wales containing many complex tables and indices. It is, however, readable and instructive. It defines adequately for future studies, stillbirths, neonatal and postnatal deaths. The author stresses the great part social, economic and environmental circumstances play in stillbirth rates. EDW. F. TARTAGLIONE, M.D.

CHILD TREATMENT AND THE THERAPY OF PLAY. By Lydia Jackson, B. Sc. and Kathleen M. Todd, M.B., D.P.M. 2nd Ed. Cloth. Price $2.50. New York: The Ronald Press, 1950.

Play therapy in this country has had diverse meanings from simple unrestricted play to highly interpretive psychoanalysis. Following a brief discussion of theories of play and how parental behavior can induce conflicts in the child, the authors describe how a child's play activities can be used to diagnose and treat these conflicts. Their rationale and approach are eclectic—with its basis in Freud, Adler and Jung. They describe, in realistic detail, how social worker and psychologist can cooperate in therapy. They are modest in their claims, admitting that the child's temperament, disposition and inheritance of physical and emotional qualities can be modified by education and psychotherapy but cannot be altered essentially. They also state that not all children should be subjected to deep therapy and name the requirements for it—a stable make-up, intelligence, and insight involving intuition and self awareness. Their treatment aims at discovering the child's own optimism within the limits of his inborn equipment—intellectual and emotional. They also emphasize the importance of work with those who control the child's life. The book is clearly and succinctly written, and devoid of technical expressions. It describes, with understanding realism, children's behavior with which every-

one who works with children is familiar. Their interpretations are convincingly valid, probably because they are delayed until there is sufficient basis for their formulation. Pediatricians will find in this book a simple, direct and lucid explanation of the many child behavior problems seen in their everyday practice. The doctor in rural sections, where child guidance clinics are not available, will find stimulus for ideas of simple ways in which he can help parents and children in their joint efforts to resolve their conflicts and to attain more effective modes of behavior.

HELEN THOMPSON, PH. D.

HANDBOOK OF ORTHOPAEDIC SURGERY. By Alfred R. Shands, Jr., M.D. in Collaboration with Richard B. Raney, M.D. 3rd Ed., Cloth. Pp. 574. Illustrated. Price $6.00. St. Louis: C. V. Mosby Company, 1948.

This book on orthopaedic surgery makes an excellent reference book for thumb nail sketches of the many common, as well as unusual conditions, which fall within the province of the subject. The clinical conditions are succinctly and orderly described and adequately illustrated by diagrams, pictures and x-ray reproductions. The book contains about 75 pages of bibliography which is an aid in obtaining more detailed information about any particular subject. This book would be of value in preparing for an examination.

MICHAEL A. BRESCIA, M.D.

THE PSYCHOLOGY OF EXCEPTIONAL CHILDREN. By Karl C. Garrison, Ph.D. Rev. Ed. Price $4.50. New York: The Ronald Press, 1950.

This book is a teaching text designed for teachers, social workers and parents. It discusses the mentally retarded, the gifted, the physically handicapped, and the emotionally and socially maladjusted child in terms of their educational problems. Parents of exceptional children continually seek the advice of their physician about the problems that they encounter in discipline and in schooling. Pediatrists or physicians will find in this book a clear discussion of how such problems may be met and how, not only the parents, but school principals and teachers may be advised in

helping these children find their optimum mental development in a society geared to the average child. Although the medical aspects may be described in an unsatisfactory manner for the medically trained, the educational problems are clearly defined and discussed.

HELEN THOMPSON, PH.D.

CROSS INFECTION IN CHILDREN'S WARDS. (British Medical Journal, London, 2:616, Sept. 17, 1949). According to Watkins and Lewis-Fanning the British Pediatric Association began in 1945 to interest itself in the problem of cross infection in children's wards in hospitals. The term cross infection was applied to any infection acquired by a patient in the hospital environment. Clinically it is an infection arising during the course of another illness, for which the patient was originally hospitalized, and may attack the respiratory tract, gastrointestinal tract, wound, skin or mucous membrane or be manifested as one of the specific fevers. Fourteen hospitals were selected as representing a cross section of children's wards in which patients were under the clinical charge of an experienced pediatrician. Children's wards in a general hospital, wards in hospitals for children only, wards in teaching and nonteaching hospitals and wards taking long-stay cases were included. In all, the records of 26 different wards were utilized. The over-all incidence of cross infection in 9,619 admissions was 7.1 per cent. The average age length of increased hospital stay of the patients that developed a cross infection was twelve days. Death was attributable in 41 children partly or wholly to cross infection. An index for the rate of cross infection was calculated for each ward (standard cross infection ratio) and was found to vary widely for the different wards even within the same hospital. Attempts were made to correlate factors which might be related to the variations, which included ward architecture, equipment, nursing, staffing visitors, type of case admitted and the age of the children. The only significant finding was that cross infection was more common in wards which admitted infants.—*Journal A.M.A.*

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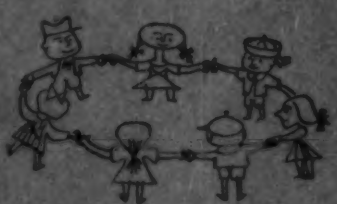
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